Pathophysiology of variable and late decelerations

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I Introduction

The classification of the fetal heart rate patterns developed around 1960 lack still a clear and definitive pathophysiological base. The names given to the several decelerations suggest however that the pathophysiological pathways along which they arise are completely clarified.

Therefore we directed our research the last years to this subject. This paper is restricted to the presentation of some data concerning the simulation of variable and late decelerations in chronically instrumented sheep preparations.

Acute changes in the fetal heart rate pattern can in general and theoretically be caused by

1. direct hemodynamic changes
2. activitation of the fetal autonomic system by chemoreceptor mediated hypoxaemia and or direct fetal adrenal release of catecholamines.
3. direct myocardial depression.

Correct interpretation of changes in the fetal heart rate pattern and estimation of the fetal condition requires knowledge about the activity of each of the three factors mentioned.

II Variable decelerations

Variable decelerations are thought to be due to obstruction of the umbilical circulation (4). Simulation of variable decelerations was performed by placing a special device around the umbilical cord directly over and fixed to the fetal abdomen (3). The device consists of two separated compartments with inflatable balloons which can be inflated separately or simultaneously. Placing both umbilical arteries in one compartment and the umbilical veins in the other compartment, or one artery and one vein in each of the compartments each possible impairment of the umbilical flow can be simulated. Following recovery from anesthesia and surgical procedures the vessels were occluded during 30 seconds. Fetal femoral artery blood samples were taken five minutes before and five minutes following the occlusion as well as at the end of the 30 seconds lasting occlusion period. With this method a total number of 197 different types of umbilical cord clampings were performed. Moreover an additional number of 42 clampings were performed during pharmocological blockade of the different parts of the autonomic nervous system.

The fetal arterial pO_2 before and at the end of the occlusion period shows a significant difference in all instances.

Occlusion of both umbilical arteries results in an immediate rise of the systolic and diastolic pressure reaching a certain steady state and followed in some occlusions by a second rise especially expressed in the diastolic pressure after a variable time, however still within the 30 seconds lasting occlusion period (fig.1).

The fetal heart frequency decreases immediately within one or two heart beats to a certain level, followed by a further decrease simultaneously with the second rise in blood pressure if present.

In case of occlusion of both umbilical veins a certain delay

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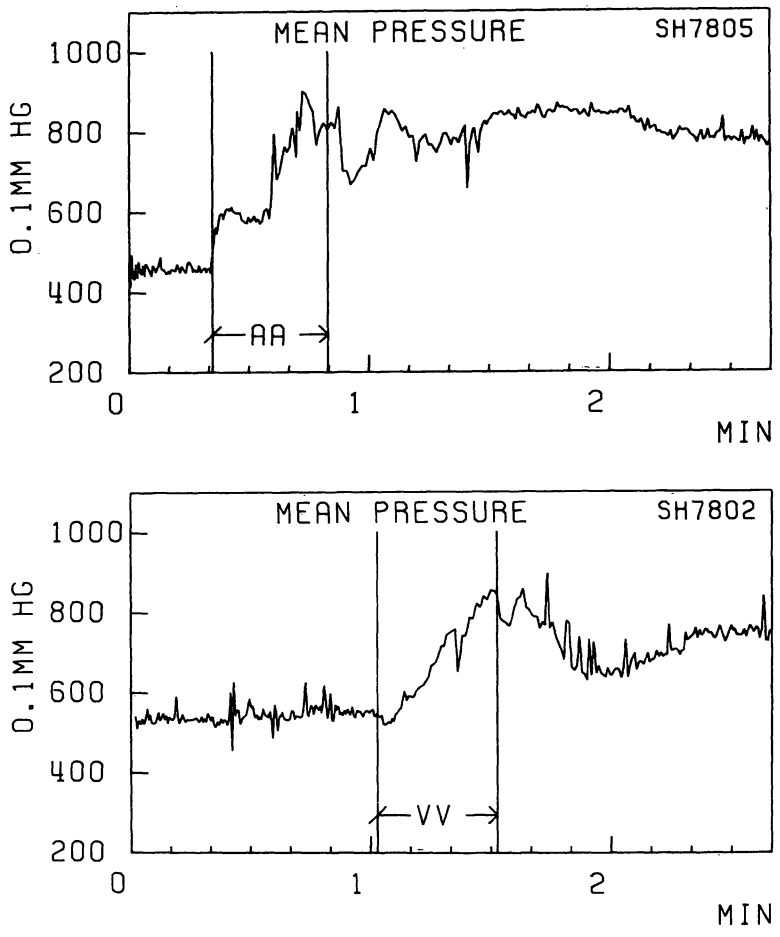

Fig. 1 Fetal systolic pressure before, during and following a 30 seconds lasting occlusion of the umbilical arteries (upper graph) and umbilical veins (lower graph). The occlusion period is indicated by the vertical bars.

is present between the start of the occlusion and the first cardiovascular reactions. The increase in systolic and diastolic blood pressure is initially preceded by a 3-5 seconds lasting decrease (fig.1). The blood pressure reaches finally the same level compared to the arterial clampings. Also during venous occlusions a second step increase in the fetal arterial blood pressure might be observed. It lasts almost ten heart beats before a decrease in the fetal heart frequency is observed in case of venous occlusions. Sometimes a small increase precedes the following decrease.

Total occlusion of the umbilical cord mimics almost completely the arterial occlusion regarding the reaction in the fetal blood pressure and heart frequency (fig.2).

The reaction in systolic and diastolic blood pressure after cholinergic blockade were in all types of occlusion completely comparable to the occlusions in fetal lambs with an intact autonomic nervous system, including the second step rise in blood pressure.

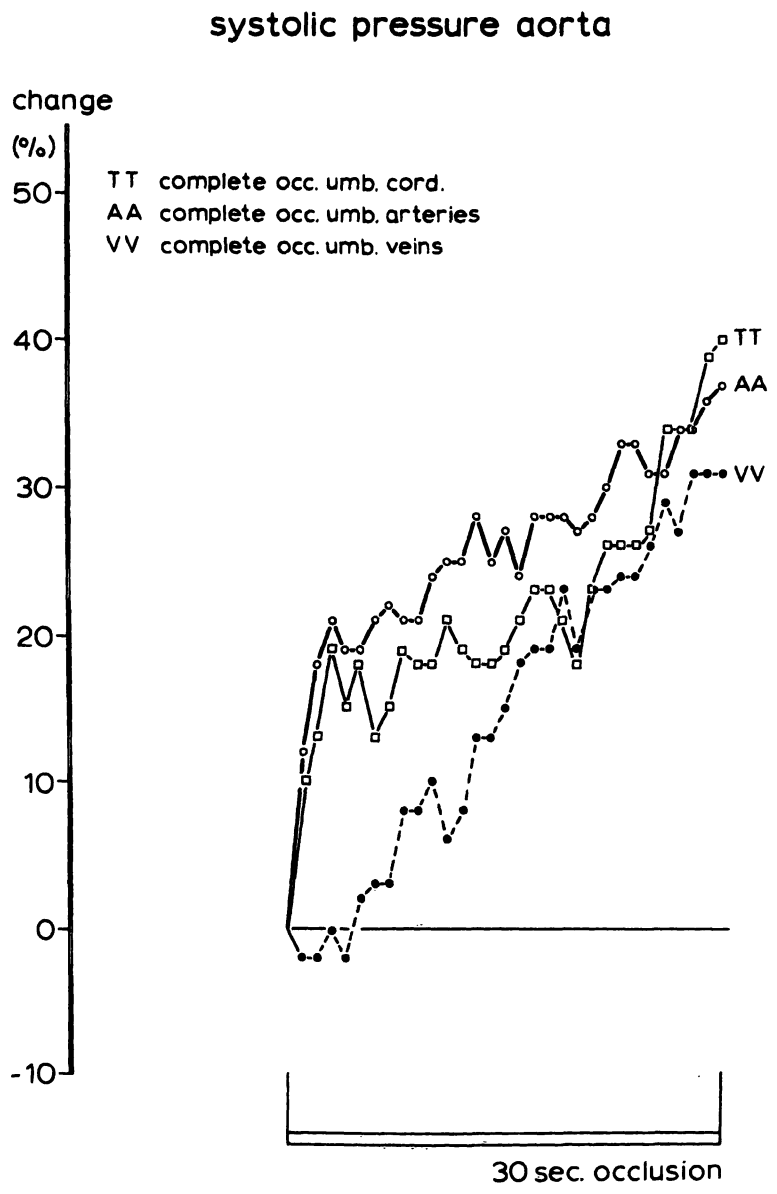

Fig. 2 Percentual change in fetal systolic pressure during 30 seconds lasting clampings of the total umbilical cord (TT), the umbilical arteries (AA) or the umbilical veins (VV).

In 11 out of 19 experiments the fetal heart frequency did not change, regardless of the kind of occlusion during cholinergic blockade (fig. 3). In six experiments a small decrease in fetal heart frequency was observed and in two experiments an increase in heart frequency was observed in occlusions during cholinergic blockade.

In case of alpha-adrenergic blockade arterial pressure initially increased followed by a decrease to values equal to or even below the pressure values before the occlusion (fig. 4). The fetal heart frequency decreased in all types of occlusion during alpha-adrenergic blockade.

The first direct increase in arterial blood pressure following occlusion of the umbilical vessels must be due to the strong increase

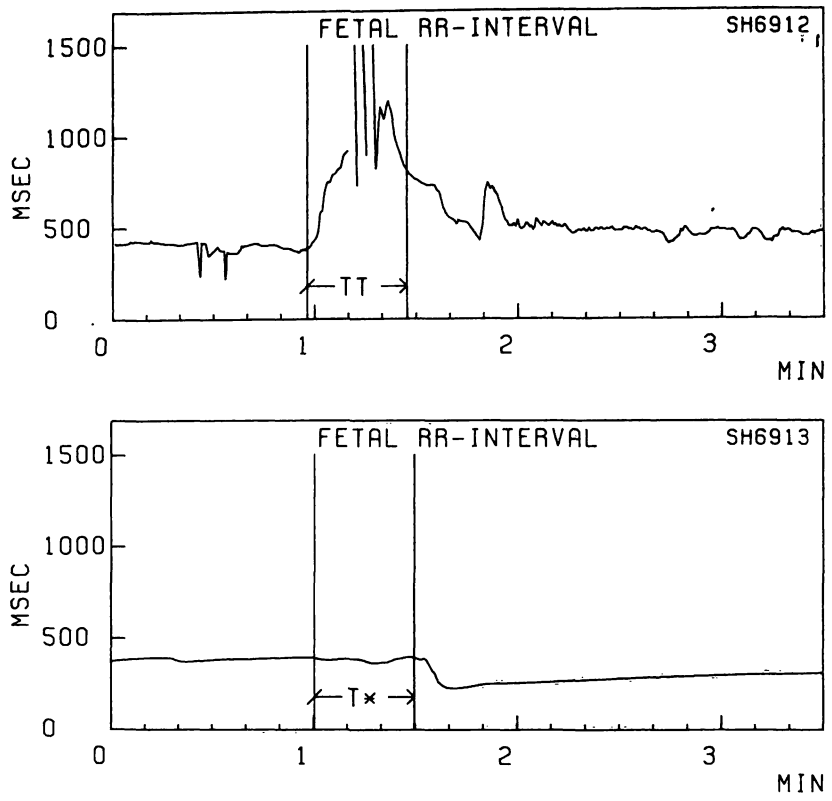

Fig. 3 Fetal R-R interval before, during and following a thirty seconds lasting occlusion of the total umbilical cord before (TT, upper graph) and after cholinergic blockade (T*, lower graph).

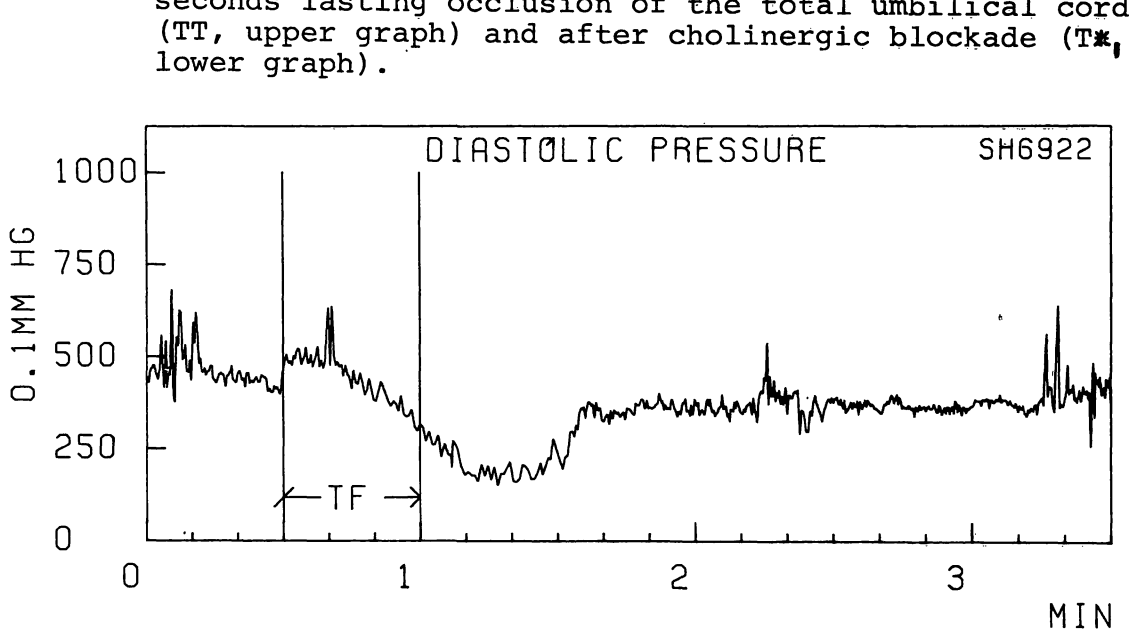

Fig. 4 Diastolic pressure before, during and following a 30 seconds lasting occlusion of the total umbilical cord during fetal alpha-adrenergic blockade. The occlusion is indicated by two vertical bars.

of the peripheral resistance by occluding the input of the placental circulation.
The baroreceptor reflex mediates the decrease in fetal heart frequency which can be concluded from occlusions during cholinergic blockade.

The second increase in blood pressure must probably be due to peripheral vasoconstriction in the course of redistribution of blood flow, caused by the decrease in partial oxygen pressure, since the fetal arterial partial oxygen pressure decreases with 20-40 percent during the occlusion. This possible mechanism is also underlined by the fact that the second arterial pressure increase is not present at all in the occlusions during alpha-adrenergic blockade.

The initial decrease in arterial pressure following venous occlusions must be caused by an initial decreased filling of the fetal heart due to trapping of a certain amount of blood in the placental circulation untill arterial pressure is maintained in the rest of the circulation. If the placental circulation would be a rigid system the reaction in the pressure parameters following an occlusion would be the same in case of compression of the umbilical arteries or the umbilical veins. Since differences as shown are present, the umbilical placental circulation must have a certain compliance which is underlined by flow measurements in one umbilical artery during occlusion of the umbilical veins.

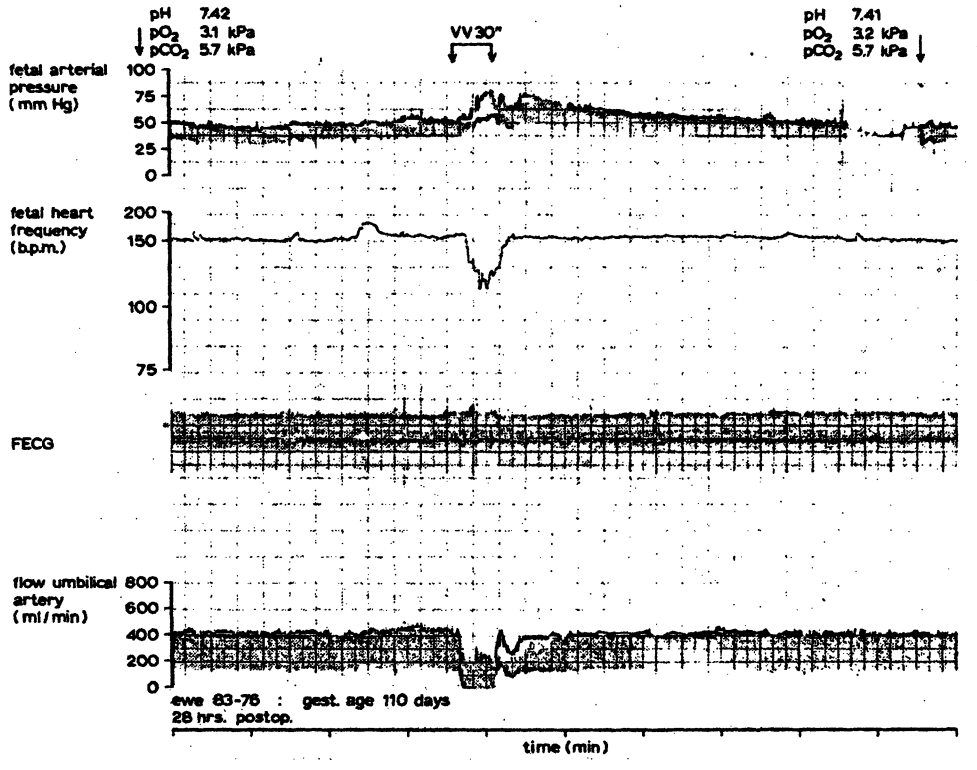

Fig. 5 Fetal arterial pressure, heart frequency, electrocardiogram and umbilical artery blood flow before, during and after a thirty seconds lasting occlusion of both umbilical veins. A positive mean arterial blood flow is present during the first seconds of the occlusion.

During several seconds following the start of the occlusion of the umbilical veins, a positive decreasing mean flow can be measured in the umbilical artery (fig.5). This lasts probably untill the system is completely filled and arterial pressure starts to rise and fetal heart frequency starts to decrease.

The degree of hypoxaemia reached in these experiments is certainly deep enough to elicit fetal adrenal catecholamine release (1,5). Since we did not measure the fetal catecholamine levels and or the velocity of fetal adrenal catecholamine relaese, it remains unclear to what extent this mechanism act upon these experiments.
Changes in fetal blood pressure and heart frequency and especially in the fetal pre-ejection time period, stronlgy suggest that this mechanism is active (2).

It is unlikely that the brief exposure to low pO_2 values in these experiments interferes with myocardial contractility.

III Late decelerations

Late decelerations are thought to be due to temporary insufficient fetal oxygen supply caused by a compromised maternal fetal exchange system.

Simulation of late decelerations was performed by placing an inflatable occluder around the common internal iliac artery. This vessel is the main uterine blood supply in the pregnant ewe. The effect of uterine contractions was simulated by periodic occlusion of this vessel for periods of from 30-60 seconds and was repeated at an interval of 2,5 minutes from the beginning of one occlusion to the start of the following compression.
To increase the degree of hypoxaemia in some experiments, the vessel was left partially occluded between the total occlusions. The experiments were performed under chronic conditions 2-9 days following surgery.

Since the effect of uterine contractions is reducing maternal placental blood flow and thus reducing oxygen delivery to the fetus, this method, causing late decelerations also, is with great certainty equivalent in its pathophysiologic mechanism to spontaneous occurring late decelerations.

At regular intervals fetal blood samples were taken. The role of reflex mechanisms in the cardiovascular changes caused by the occlusions was studied by blocking parts of the autonomic nervous system pharmacologically.

Periodic occlusion of the common internal iliac artery results during the first occlusions either in a fetal heart frequency deceleration or an acceleration. From the fourth occlusion onwards almost always a deceleration is present (fig.6). The time relationship between the increase in fetal blood pressure and decrease in fetal heart frequency varied. In some instances the heart frequency started to decrease several seconds before the onset of blood pressure increase. In non acidemic fetuses the highest blood pressure and the lowest fetal heart rate occurred generally during the same time period.
With progressing fetal acidosis a complementary fetal tachycardia occurred and the amount of deceleration was less, whereas the pattern of progressive hypertension disappeared. Below a fetal arterial pH of 7.10 a decrease in blood pressure accompanied the fetal heart rate deceleration (fig.7).

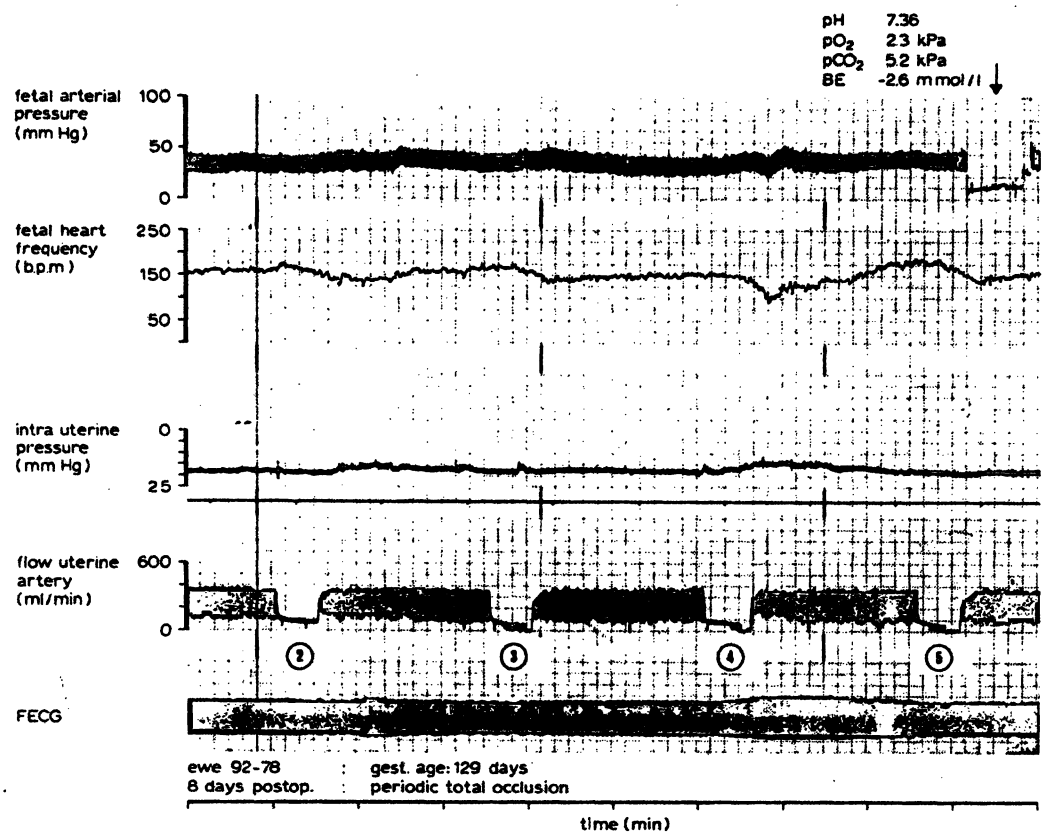

Fig. 6 Periodic total occlusion of the common internal iliac artery. The figures below the uterine blood flow tracing indicate the number of occlusion.

Alpha-adrenergic blockade with phentolamine prevented the progressive hypertensive response of the fetus to the periodic interruption of the uterine blood flow. Some cardiac slowing could be present however in occlusions during alpha-adrenergic blockade (fig.8).

During cholinergic blockade with atropine, periodic accelerations occurred in response to the occlusions in case of non acidemic fetuses. These accelerations could be eliminated by beta-adrenergic blockade with propranolol, but not with an alpha-adrenergic blocking agent.

Administration of atropine to the already acidotic fetus resulted in an increase in the base line fetal heart rate, but no change in the amount of the deceleration occurred (fig.9).

The beat to beat variability of the baseline fetal heart rate as judged visually persisted and disappeared very lately in case of severe fetal acidemia.

The observations just described suggest several reflex pathways in producing late decelerations (fig.10). The dominant reflex is the chemoreceptor mediated reflex vasoconstriction, producing an increase in blood pressure and in turn baro-reflex induced cardiac slowing. Direct stimulation by hypoxaemia of the cardiodecelerator reflex is probably also possible since the onset of the deceleration can precede the increase in blood pressure. Moreover cardiac slowing is present in occlusions during alpha-adrenergic blockade.

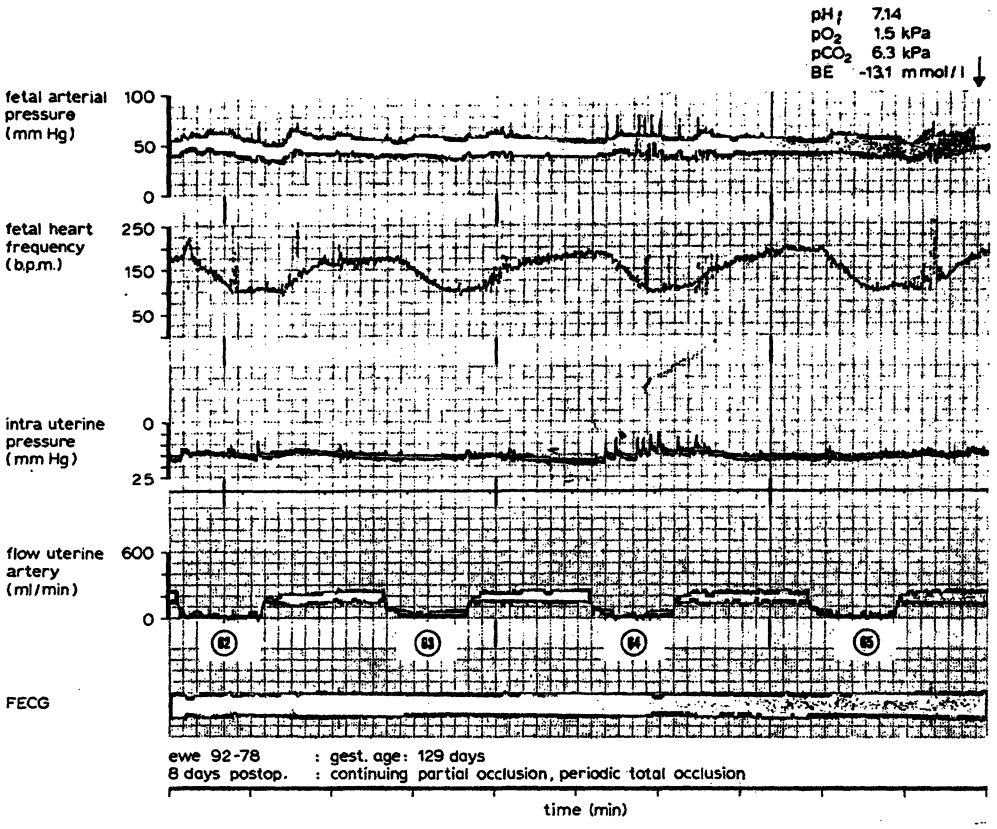

Fig. 7 Continuing partial occlusion with periodic total occlusion of the common internal iliac artery. The figures below the uterine blood flow tracing indicate the number of occlusion. In this acidotic fetus arterial blood pressure decreases during the fetal heart rate decelerations.

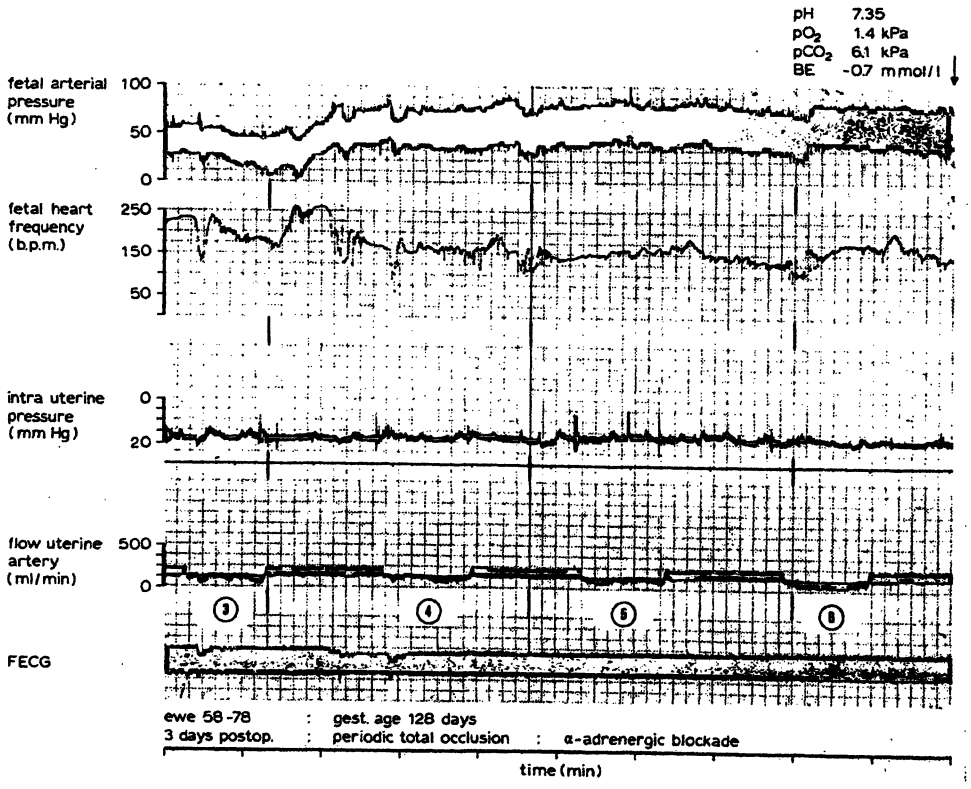

Fig. 8 Periodic total occlusion of the common internal iliac artery after blocking the fetal alpha-adrenergic system.

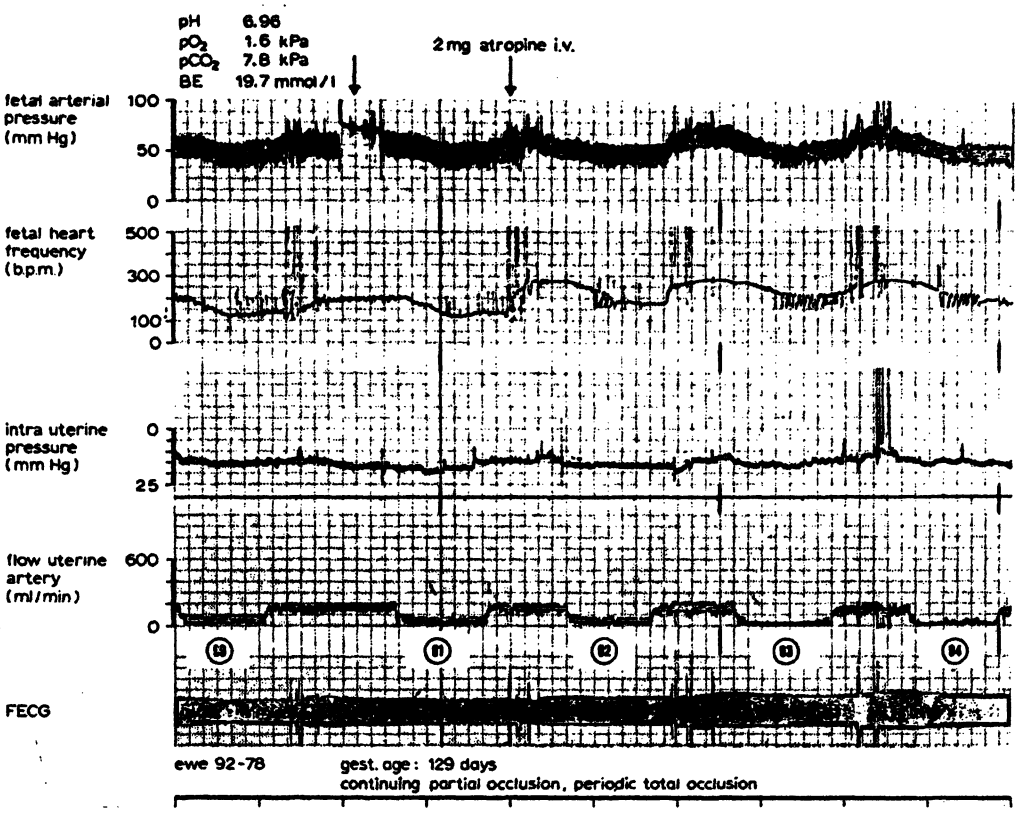

Fig. 9 Continuing partial occlusion with periodic total occlusion of the common internal iliac artery before and following fetal cholinergic blockade in case of a severe distressed fetus.

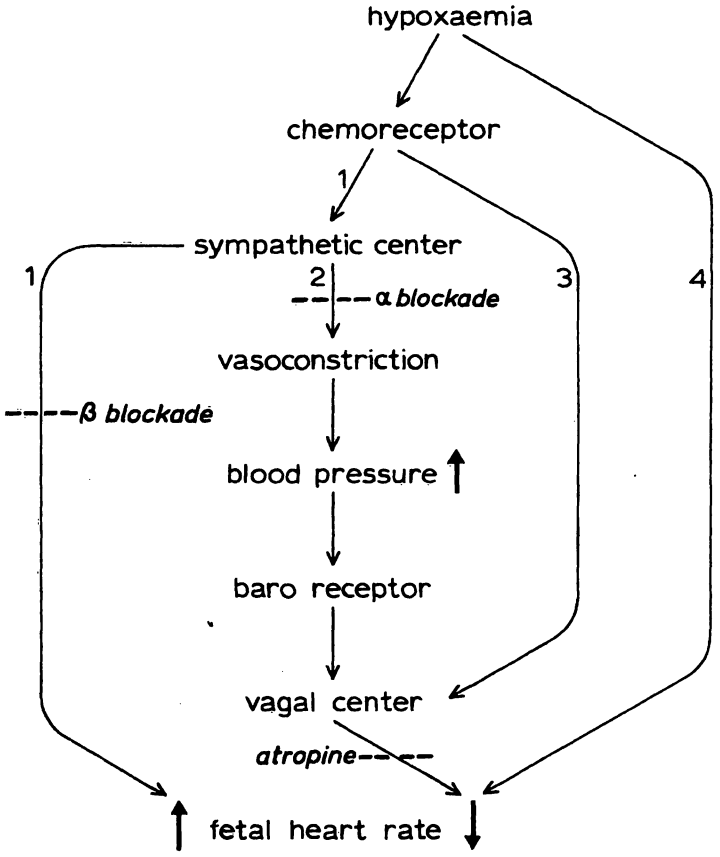

Fig. 10 Possible pathways producing periodic changes in the fetal heart rate pattern due to hypoxaemia - hypoxia.

An adrenergic cardioaccelerator component is also present as demonstrated from the accelerations in occlusions during cholinergic blockade and from the accelerations during the very first occlusions in the experiment. This accelerations could be blocked by propranolol.

The last pathway is the direct hypoxic depression of the fetal myocardium under conditions of severe fetal acidosis as shown from the experiments under cholinergic blockade and severe fetal acidosis. The exact point at which reflex mechanisms in producing late decelerations are replaced by direct hypoxic depression is however not determined. Therefore more experiments has to be performed, although this transient point seems to lie somewhere around a pH of 7.25. This point is probably different in each fetus dependent also on the previous oxygen content, cardiac glycogen stores and blood glucose levels.

Although results obtained in animal experiments can never be transposed completely on human fetuses, with these data several phenomena in human fetal heart rate patterns can be understood better.

REFERENCES

1. Comline, R.S., Silver, I.A., Silver, M. : Factors responsible for the stimulation of the adrenal medulla during asphyxia in the fetal lamb. J. Physiol. 178 : 211, 1965.

2. Evers, J.L.H.: The cardiac pre-ejection period during prenatal life. Studies in stressed and unstressed fetal lambs. Thesis, Catholic University, Nijmegen 1978.

3. de Haan, J., Jongsma, H.W., Crevels, A.J., Arts, T.H.M. : The cardiovascular effects following compression of the umbilical veins and or arteries in chronic sheep preparation. In : Abstracts of free communications. 5th European Congress of Perinatal Medicine p. 50, nr 79. Almqvist and Wiksell Intern. Stockholm 1976.

4. Hon, E.H. : An atlas of fetal heart rate patterns. Harty Press Inc., New Haven 1968.

5. Jones, C.T., Robinson, R.D. : Plasma catecholamines in foetal and adult sheep. J. Physiol. 248 : 15, 1975

6. Martin, C.B., de Haan, J., van der Wildt, B., Jongsma, H.W., Dieleman, A., Arts, T.H.M. : Mechanisms of late decelerations in the fetal heart rate; A study with autonomic blocking agents in fetal lambs (in press).

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